The correspondence section is a public forum and, as such, is not peer-reviewed. EHP *is not responsible for the accuracy, currency, or reliability of personal opinion expressed herein; it is the sole responsibility of the authors.* EHP *neither endorses nor disputes their published commentary.*

Nanomaterials and the Precautionary Principle

doi:10.1289/ehp.1103687

Kessler (2011) provided a valuable update on the current state of research and regulatory policy concerning nanomaterials. However, the article could give the misleading impression that the precautionary principle constitutes a straightforward guideline for improving public policy in this area. Instead, the precautionary principle provides only a general framework that must be specified before one can adequately assess its implications for policy.

Near the beginning of the article, Kessler (2011) quoted Alexis Baden-Mayer, who worried,

> [I]n our regulation of food and consumer products, we don't implement the precautionary principle. Things go to market before we know whether or not they're really safe for human beings over the long term.

Kessler (2011) concluded with a quotation from Michael Hansen:

> I think we need to take a precautionary approach because we've learned the hard way over and over and over again. You'd think we would learn.

By framing the issues in this way, Kessler (2011) intimated that the precautionary principle could serve as a valuable guide for future research and policy making. However, without further specification, the principle provides only a rough outlook or orientation rather than a specific regulatory plan of action; its merits cannot be clearly evaluated unless a number of further questions are answered.

A number of scholars have attempted to clarify how various formulations of the precautionary principle relate to one another. There are at least three important features that vary in different accounts of the principle: *a*) the threats that ought to be addressed; *b*) the amount and kinds of knowledge necessary to justify precautionary measures; and *c*) the specific precautionary measures that ought to be taken (Elliott 2010; Manson 2002; Sandin 1999). All three issues require further discussion in the case of nanomaterial research and regulation.

Regarding threats, one of the most crucial issues is whether it is sufficient to show that nanoparticles are safe for humans or whether they must also be shown to be safe for the environment—and, if so, what environmental impacts must be tested. Andrew Maynard hinted at this issue:

> I think there is a greater chance that we're going to see long-term environmental impacts from these materials than we are going to see short-term consumer impacts. (Kessler 2011)

Given the vast array of nanoparticles under consideration, it seems doubtful that they could all be thoroughly tested for a wide range of environmental effects before allowing their use.

This raises the question of how much evidence should be demanded before approving particular sorts of nanoparticles. A number of questions are relevant here, some of which are touched on by Kessler (2011): What kinds of screening studies should be required? When should *in vivo* studies be required? What structural or functional changes to a nanoparticle (e.g., size, crystal structure, manufacturing process) should trigger new toxicity studies? Should by-products of the production process also be studied in order to declare a nanoparticle safe (Templeton et al. 2006)? What steps must be taken to ensure that multiple manufacturing batches of the same nanoparticle result in products with the same toxicity profile? Does it matter what kinds of consumer products the nanoparticles are used for?

Finally, although many proponents and opponents of the precautionary principle treat the precautionary principle as if it requires bans on potential threats until they are shown to be safe, a range of other positions are also available on this issue. Three options include *a*) insisting that government agencies be notified when products contain particular nanoparticles; *b*) demanding labeling; or *c*) taking steps to minimize human or environmental exposure to nanoparticles until they have received further testing. Kessler (2011) highlighted our present failure to achieve some of these minimal steps.

These considerations do not by themselves count as sufficient reasons for rejecting the precautionary principle, but they do show that the decision to adopt it is the start of a complicated conversation rather than a straightforward choice about how to regulate nanomaterials.

This work was supported in part by grant 0809470 from the National Science Foundation, NSF 06-595, to the Nanotechnology Interdisciplinary Research Team (NIRT): Intuitive Toxicology and Public Engagement.

The author declares he has no actual or potential competing financial interests.

Kevin C. Elliott
Department of Philosophy and
USC NanoCenter
University of South Carolina
Columbia, South Carolina
E-mail: ke@sc.edu

References

Elliott K. 2010. Geoengineering and the precautionary principle. Int J Appl Phil 24:237–253.

Kessler R. 2011. Engineered nanoparticles in consumer products: understanding a new ingredient. Environ Health Perspect 119:A120–A125.

Manson N. 2002. Formulating the precautionary principle. Environ Ethics 24:263–274.

Sandin P. 1999. Dimensions of the precautionary principle. Human Ecol Risk Assess 5:889–907.

Templeton RC, Ferguson PL, Washburn KM, Scrivens WA, Chandler GT. 2006. Life-cycle effects of single-walled carbon nanotubes (SWNT) on an estuarine meiobenthic copepod. Environ Sci Technol 40:7387–7393.

Manganese in Drinking Water and Intellectual Impairment in School-Age Children

doi:10.1289/ehp.1103485

We read with interest the the article by Bouchard et al. (2011) on the effect of manganese in drinking water on children's IQ (intelligence quotient). In this cross-sectional study, the authors examined IQ scores in relation to manganese exposure using four exposure metrics: *a*) concentration of manganese in tap water; *b*) concentration of manganese in hair samples; *c*) estimate of manganese intake from water consumption; and *e*) estimate of manganese intake from diet consumption.

One key finding from the study of Bouchard et al. (2011) is that a higher concentration of manganese in tap water was significantly associated with lower IQ. Compared with the other three exposure metrics used in the study, the concentration of manganese in water followed an almost perfect dose–response relationship with children's IQ, and it was shown to be a better predictor of lower IQ than the exposure metrics. We found this surprising for three reasons. First, in their analysis of the association between concentration of manganese in tap water and IQ, Bouchard et al. included the entire study population (n = 362). We consider this inappropriate because 33% of the study participants (n = 121) did not drink tap water at home. Thus, these 121 children may have experienced much lower exposure to manganese from tap water than the remaining children in the study. Second, if we consider the highest quintile of water-manganese concentration (median, 216 μg/L), the estimated manganese intake from water would be ≤ 0.43 mg/day for half of the children in this exposure group, assuming a daily water intake of 2 L. Even at this level, the intake of manganese from water was still far below the daily intake recommended by the Institute of Medicine (2001): children 1–3 years of age (1.2 mg/day) and children 4–13 years of age (1.5–1.9 mg/day). Third, Bouchard et al. reported that the children's manganese intake from food was more than two orders of magnitude compared to

the amount ingested from water. This suggests that if elevated manganese was causally related to lower IQ, the decrease in IQ was more likely due to the intake of manganese from both water and food sources than from water alone. While one can postulate differences in bioavailability between manganese in food and in water, these would need to be considerable to result in equal or greater uptake from water than from food.

The utility of hair as a biomarker for human exposure to manganese has yet not been established [Agency for Toxic Substances and Disease Registry (ATSDR) 2001]. There is still a lack of standard procedure for collection of hair samples as well as insufficient evidence to demonstrate the effect of washing hair on analytical results (ATSDR 2001). Bouchard et al. (2011) excluded children with dyed hair, but it would be interesting to also distinguish children with natural hair of different colors in the analysis, because levels of manganese in hair can vary by natural colors of hair.

Bouchard et al. (2011) generated an interesting hypothesis on neurotoxicity of water manganese in children at a level that is currently considered to have no adverse effect (World Health Organization 2008), but we believe more studies will be needed to confirm their findings. To better characterize human exposure to manganese from water, it is important for future studies to quantify bioavailability of manganese from water and from food sources. In addition, employing a prospective study design and controlling for all possible risk factors—including overall nutritional status—will be critical. Additionally, comparing hair with other biomarkers of manganese exposure would be another area to explore for future studies.

The authors declare they have no actual or potential competing financial interests.

Hong Chen
Ray Copes
Ontario Agency for Health Protection and Promotion
Toronto, Ontario, Canada
E-mail: hong.chen@ohapp.ca

References

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. Summary Report Hair Analysis Panel Discussion Exploring the State of the Science. Available: http://www.atsdr.cdc.gov/HAC/hair_analysis/index.html [accessed 23 December 2010].

Bouchard MF, Sauvé S, Barbeau B, Legrand M, Brodeur M, Bouffard T, et al. 2011. Intellectual impairment in school-age children exposed to manganese from drinking water. Environ Health Perspect 119:138–143.

Institute of Medicine. 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium and Zinc. Washington, DC:National Academy Press. Available: http://www.nap.edu/catalog.php?record_id=10026 [accessed 23 December 2010].

World Health Organization. 2008. Chemical aspects. In: Guidelines for Drinking-water Quality: Third Edition Incorporating the First and Second Addenda, Vol 1: Recommendations. Geneva:World Health Organization, 145–196g. Available: http://www.who.int/water_sanitation_health/dwq/gdwq3rev/en/ [accessed 10 May 2011].

Manganese in Drinking Water: Bouchard Responds

doi:10.1289/ehp.1103485R

Chen and Copes raise some interesting issues regarding our article (Bouchard et al. 2011). In our study we investigated the change in IQ scores with respect to different exposure metrics for manganese. One of these metrics was home tap water manganese concentration, which was strongly associated with IQ deficits. Chen and Copes indicate that they consider it inappropriate to include in this analysis children who did not drink tap water at home. Second, they note that even for children in the highest quintile of water manganese concentration, the intake of manganese from water ingestion is below the recommended dietary manganese intake (Institute of Medicine 2001). In response to their first point, it is important to consider that children who do not drink tap water are still exposed through the consumption of many foods and drinks prepared with tap water. In addition, and perhaps most important, children might be exposed by inhalation of aerosols containing manganese when showering (Elsner and Spangler 2005). If this represents a significant source of exposure, which is unclear (Aschner 2006; Spangler and Elsner 2006), inhalation of aerosols could be responsible for inducing neurotoxic effects. Indeed, inhaled manganese is delivered to the brain much more efficiently than ingested manganese, because it bypasses normal homeostatic mechanisms.

Third, Chen and Copes make the point that because dietary intake of manganese intake is much higher than the amount ingested from water, the decrease in IQ is more likely due to intake of manganese from water and food sources collectively, rather than from water alone. The intake of manganese from water consumption was indeed very small compared with dietary intake (medians, 8 and 2,335 μg/kg/month, respectively), but we found no evidence that dietary manganese is related to cognitive abilities. As we reported in our article (Bouchard et al. 2011), dietary manganese intake, assessed with a food frequency questionnaire, was not associated with IQ and did change the point estimates for water manganese concentration when included in the regression model.

We believe that the interpretations that assimilate manganese present in water to dietary manganese have had the effect of dismissing the potential risks of this source of exposure, thus slowing research into this question. Little is known about the absorption and retention of manganese from food versus water, or about inhalation of aerosols in showers. Although more research is necessary to understand the mechanisms by which manganese present in water might be neurotoxic for children, we believe that our findings offer strong support for this hypothesis. Because manganese levels associated with significant cognitive deficits in our study are common in groundwater, this problem could have a great public health importance. For instance, 11% of domestic wells have manganese concentrations > 140 μg/L in the United States (U.S. Geological Survey 2009). We agree that additional studies, ideally with a prospective design, are necessary.

Finally, a valid biomarker of manganese exposure would greatly advance our understanding of this metal's toxic effects. We used hair, notably because its collection is much less invasive than blood sampling. Chen and Copes rightly point out the limitations of hair as a biomarker, and research should explore new biomarkers. For instance, in a small study, saliva manganese levels were significantly higher in welders than in nonexposed subjects, and levels increased in welders with the more years of exposure (Wang et al. 2008). Also in that study, saliva manganese concentrations correlated with serum concentrations. Saliva is less invasive to collect than blood and less prone to external contamination than hair; thus, it might be a useful biomarker.

The author declares that she has no actual or potential competing financial interests.

Maryse F. Bouchard
Department of Environmental and Occupational Health
University of Montreal
Montreal, Québec, Canada
E-mail: maryse.bouchard@umontreal.ca

References

Aschner M. 2006. Manganese in the shower: mere speculation over an invalidated public health danger. Med Hypotheses 66(1):200–201.

Bouchard MF, Sauvé S, Barbeau B, Legrand M, Brodeur M, Bouffard T, et al. 2011. Intellectual impairment in school-age children exposed to manganese from drinking water. Environ Health Perspect 119:138–143.

Elsner RJ, Spangler JG. 2005. Neurotoxicity of inhaled manganese: public health danger in the shower? Med Hypotheses 65(3):607–616.

Institute of Medicine. 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium and Zinc. Washington, DC:National Academy Press. Available: http://www.nap.edu/catalog.php?record_id=10026 [accessed 16 February 2011].

Spangler JG, Elsner R. 2006. Commentary on possible manganese toxicity from showering: response to critique. Med Hypotheses 66(6):1231–1233.

U.S. Geological Survey. 2009. Quality of Water from Domestic Wells in the United States. Available: http://water.usgs.gov/nawqa/studies/domestic_wells [accessed 16 February 2011].

Wang D, Du X, Zheng W. 2008. Alteration of saliva and serum concentrations of manganese, copper, zinc, cadmium and lead among career welders. Toxicol Lett 176(1):40–47.

ERRATUM: Assessing Long-Term Exposure in the California Teachers Study

In an article published in *Environmental Health Perspectives* (Ostro et al. 2010), we analyzed the relationships of long-term exposure to fine particulate matter (≤ 2.5 μm in aerodynamic diameter; $PM_{2.5}$) and its components with mortality in a cohort of > 100,000 active and retired female professionals participating in the California Teachers Study (CTS) cohort. We used a Cox proportional hazards model in which pollution exposure was measured as a continuous variable over the study period. Monthly average pollutant concentrations were obtained for each participant from measurements at the nearest $PM_{2.5}$ monitor within either 8 or 30 km of her geocoded residential address. Each participant was assigned a single exposure value over the follow-up period, defined as the average pollutant concentration from the beginning of the observation period (1 June 2002) to the woman's date of death, loss to follow-up, or study termination (31 July 2007). Thus, exposure assignment was dependent on the duration of follow-up for each participant.

In our article (Ostro et al. 2010), we reported associations of mortality from all causes, cardiopulmonary disease, and ischemic heart disease (IHD) with $PM_{2.5}$ mass and several of its components. However, the estimated hazard ratios (HRs) were generally higher than those reported from previous cohort studies (Dockery et al. 1993; Eftim et al. 2008; Krewski et al. 2009; Laden et al. 2006; Pope et al. 1995). Part of this difference was likely due to the nature of the exposure assignment. Most previous cohort studies have assigned the same exposure period to all study subjects, regardless of when deaths occurred. Thus, estimated exposures for some study participants in several studies occurred after their deaths. In addition, exposures have usually been assigned to participants based on their residential address at enrollment only, without taking into account exposure changes that may have occurred throughout the study period or when participants relocated. Finally, many previous studies measured exposure for only a subset of the years during which the cohort was followed. In an effort to reduce these aspects of exposure misclassification, we estimated exposures beginning prior to the cohort follow-up period, continuing to the end of the study or until the participant died or relocated out of state, incorporating updated exposure assignments when the subjects moved.

Importantly, measured concentrations of several pollutants in California declined substantially from 2002 through 2007; annual average $PM_{2.5}$, organic carbon (OC), and nitrates decreased by around 30% each. These marked decreases in ambient $PM_{2.5}$ concentrations resulted in lower average exposure estimates for cohort members who survived to the end of our study. Thus, the exposure assigned to a participant who died at time *t* would tend to be greater for events occurring early in the observation period, compared with the long-term average exposures of the participants who comprised the remainder of the risk set (i.e., those who were still part of the cohort study at time *t* and who subsequently experienced lower ambient pollution levels).

We have reanalyzed the CTS data using time-dependent pollution metrics—in which the exposure estimates for everyone remaining alive in the risk set were recalculated at the time of each death—in order to compare their average exposures up to that time with that of the individual who had died. In this way, decedents and survivors comprising the risk set had similar periods of pollution exposure, without subsequent pollution trends influencing the surviving women's exposure estimates.

As in our previous study (Ostro et al. 2010), we restricted the sample in this reanalysis to women living within 30 km of one of eight fixed-site monitors in the U.S. Environmental Protection Agency's Speciation Trend Network (STN), resulting in a study population of almost 44,000 women. Residential addresses from study enrollment forward were geocoded and linked with monthly pollutant averages at the nearest STN monitor to generate estimates of long-term exposure. We also used the same set of individual and ecological covariates in a Cox proportional hazards model as was used in the original study. Pollutants entered separately into the model included $PM_{2.5}$ mass, elemental carbon (EC), OC, sulfate, nitrate, iron, potassium, silicon, and zinc. We used data on primary cause of death from August 2002 through July 2007 to examine the relationships between pollutants and mortality from all causes and cardiopulmonary, pulmonary, and IHDs.

The results are summarized in Erratum Table 1, scaled to the interquartile range (IQR) for each pollutant. HRs were significantly attenuated from our previous results. No associations were observed between all-cause mortality and $PM_{2.5}$ or its components. For cardiopulmonary mortality, we observed significant associations for $PM_{2.5}$ mass, nitrate, sulfate, and silicon, with more modest associations for zinc. $PM_{2.5}$ mass and all of its components were associated with mortality from IHD, whereas none of the pollutants was associated with

Erratum Table 1. Association between mortality outcomes and $PM_{2.5}$ and its components using a 30-km buffer (n = 43,220).

		All-cause (ICD-10 codes, all except S–Z) (n = 2,519)		Cardiopulmonary (ICD-10 codes, I00–I99, J00–J98) (n = 1,357)		Ischemic heart disease (ICD-10 codes, I20–I25) (n = 460)		Pulmonary (ICD-10, codes J00–J98) (n = 355)	
Pollutant	IQR ($\mu g/m^3$)	HR (95% CI)	*p*-Value	HR (95% CI)	*p*-Value	HR (95% CI)	*p*-Value	HR (95% CI)	*p*-Value
$PM_{2.5}$	6.1	1.03 (0.98–1.10)	0.26	1.11 (1.03–1.21)	0.01	1.31 (1.14–1.50)	< 0.01	1.02 (0.87–1.19)	0.84
EC	0.65	1.02 (0.93–1.12)	0.65	1.07 (0.94–1.22)	0.28	1.46 (1.17–1.83)	< 0.01	0.88 (0.68–1.15)	0.35
OC	0.84	1.00 (0.95–1.04)	0.91	1.04 (0.98–1.11)	0.19	1.13 (1.01–1.25)	0.03	0.95 (0.84–1.06)	0.35
Sulfate	2.2	1.06 (0.97–1.16)	0.18	1.14 (1.01–1.29)	0.03	1.48 (1.20–1.82)	< 0.01	1.04 (0.82–1.31)	0.77
Nitrate	3.2	1.03 (0.98–1.09)	0.27	1.11 (1.03–1.19)	0.01	1.27 (1.12–1.43)	< 0.01	1.04 (0.90–1.20)	0.58
Iron	0.13	1.01 (0.93–1.11)	0.77	1.05 (0.93–1.19)	0.40	1.39 (1.13–1.72)	< 0.01	0.88 (0.69–1.13)	0.32
Potassium	0.07	1.01 (0.94–1.08)	0.85	1.06 (0.97–1.17)	0.22	1.27 (1.07–1.49)	< 0.01	0.90 (0.74–1.09)	0.27
Silicon	0.03	1.02 (0.99–1.06)	0.22	1.05 (1.00–1.10)	0.04	1.11 (1.02–1.20)	0.01	0.98 (0.89–1.08)	0.71
Zinc	0.01	1.03 (0.96–1.11)	0.45	1.09 (0.98–1.20)	0.10	1.33 (1.12–1.58)	< 0.01	0.97 (0.79–1.18)	0.74

ICD-10, *International Classification of Diseases,* 10th Revision (World Health Organization 1993). Values shown are HRs [95% confidence intervals (CIs)] and *p*-values scaled to the IQR of each pollutant. All models are adjusted for smoking status, total pack-years, body mass index, marital status, alcohol consumption, second-hand smoke exposure at home, dietary fat, dietary fiber, dietary calories, physical activity, menopausal status, hormone replacement therapy use, family history of myocardial infarction or stroke, blood pressure medication and aspirin use, and neighborhood contextual variables (income, income inequality, education, population size, racial composition, unemployment).

pulmonary mortality. This Erratum Table 1 should replace Table 5 in our previous article (Ostro et al. 2010).

Compared with our previous results (Ostro et al. 2010), these updated $PM_{2.5}$ HRs are more consistent with several other published estimates of mortality risks, which are scaled to an increment of 10 μg/m^3 of long-term average $PM_{2.5}$ and summarized in Erratum Table 2. For example, relative to our revised HR of 1.19 for cardiopulmonary disease, analogous HRs from previous studies include 1.09 (95% CI, 1.03–1.16) from the American Cancer Society–Cancer Prevention II (ACS) cohort (cardiopulmonary disease; Pope et al. 2004), 1.28 (95% CI, 1.13–1.44) from the Harvard Six Cities study (cardiovascular disease; Laden et al. 2006), and 1.10 (95% CI, 0.94–1.28) from the Los Angeles subcohort of the ACS study (cardiopulmonary disease; Jerrett et al. 2005). Much higher HRs were observed in the observational study of the Women's Health Initiative cohort for cardiovascular and IHD mortality (Miller et al. 2007).

These revised results still support the existence of elevated risks of $PM_{2.5}$-associated cardiopulmonary disease and IHD, and illustrate the importance of considering the impact of long-term pollution trends in modeling estimates of exposure.

The authors declare they have no actual or potential competing financial interests.

Bart Ostro
California Office of Environmental Health Hazard Assessment
Oakland, California
E-mail: Bostro@Creal.cat

Peggy Reynolds
Debbie Goldberg
Andrew Hertz
Cancer Prevention Institute of California
Berkeley, California

Richard T. Burnett
Hwashin Shin
Health Canada
Ottawa, Ontario, Canada

Edward Hughes
Edward Hughes Consulting
Ottawa, Ontario, Canada

Cynthia Garcia
California Air Resources Board Sacramento, California

Katherine D. Henderson
Leslie Bernstein
City of Hope
Duarte, California

Michael Lipsett
California Department of Public Health
Richmond, California

References

Chen LH, Knutsen SF, Shavlik D, Beeson WL, Petersen F, Ghamsary M, et al. 2005. The association between fatal coronary heart disease and ambient particulate air pollution: are females at greater risk? Environ Health Perspect 113:1723–1729.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, et al. 1993. An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753–1759.

Eftim SE, Samet JM, Janes H, McDermott A, Dominici F. 2008. Fine particulate matter and mortality: a comparison of the Six Cities and American Cancer Society cohorts with a Medicare cohort. Epidemiology 19:209–216.

Jerrett M, Burnett RT, Ma R, Pope CA III, Krewski D, Newbold KB, et al. 2005. Spatial analysis of air pollution and mortality in Los Angeles. Epidemiology 16:727–736.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, et al. 2009. Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution Aand Mortality. HEI Research Report 140. Boston, MA:Health Effects Institute.

Laden F, Schwartz J, Speizer FE, Dockery DW. 2006. Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard Six Cities study. Am J Respir Crit Care Med 173:667–672.

Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, et al. 2007. Long-term exposure to air pollution and incidence of cardiovascular events in women. N Engl J Med 356:447–458.

Ostro B, Lipsett M, Reynolds P, Goldberg D, Hertz A, Garcia C, et al. 2010. Long-term exposure to constituents of fine particulate air pollution and mortality: results from the California Teachers Study. Environ Health Perspect 118:363–369.

Pope CA III, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, et al. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287:1132–1141.

Pope CA III, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, et al. 2004. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109:71–77.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, et al. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669–674.

Puett RC, Hart JE, Yanosky JD, Paciorek C, Schwartz J, Suh H, et al. 2009. Chronic fine and coarse particulate exposure, mortality and coronary heart disease in the Nurses' Health Study. Environ Health Perspect 117:1697–1701.

World Health Organization 1993. International Classification of Diseases, 10th Revision. Geneva:World Health Organization.

Erratum Table 2. Comparative HRs (95% CIs) associated with a 10-μg/m^3 change in long-term exposure to $PM_{2.5}$ in several cohort studies conducted in the United States.

Authors	Exposure assessment	All causes	Cardiovascular	Cardiopulmonary	IHD
Ostro et al. (this report)	From 1 year prior to follow-up until event (either death or end of study), time-dependent	1.06 (0.96–1.16)[a]		1.19 (1.05–1.36)[a]	1.55 (1.24–1.93)[a]
Ostro et al. (2010)	From 2 months prior to study through event month	1.84 (1.66–2.05)[a]		2.05 (1.80–2.36)[a]	2.89 (2.27–3.67)[a]
Pope et al. (2002, 2004)	Four years prior to or at start of follow-up and 2 years after end of follow-up	1.06 (1.02–1.11)	1.12 (1.08–1.15)	1.09 (1.03–1.16)	
Laden et al. (2006)	Multiyear average concurrent with follow-up	1.16 (1.07–1.26)	1.28 (1.13–1.44)		
Miller et al. (2007)	One year in middle of follow-up		1.76 (1.25–2.47)[a]		2.21 (1.17–4.16)[a]
Jerrett et al. (2005)	One year at end of follow-up	1.15 (1.03–1.29)		1.10 (0.94–1.28)	1.32 (1.05–1.66)
Eftim et al. (2008)	Three-year average concurrent with follow-up	1.21 (1.15–1.27)			
Chen et al. (2005)	Four-year moving average prior to event				1.42 (1.06–1.90)[a]
Puett et al (2009)	One year prior to event	1.26 (1.02–1.54)[a]			2.02 (1.07–3.78)[a]

HRs are scaled to 10-μg/m^3 change in $PM_{2.5}$, in contrast to Table 1, in which HRs are scaled to the pollutant interquartile range.
[a]Women only.